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s il(w)18 and alzheimer?
          581352  IL
         1842927  18
           12821  IL(W)18
          267019  ALZHEIMER?
      S6      65  IL(W)18 AND ALZHEIMER?
? rd s6
      S7      27  RD S6  (unique items)
? t s7/7/all

 7/7/1     (Item 1 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
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0021279913   BIOSIS NO.: 200900621350
Glyburide inhibits the Cryopyrin/Nalp3 inflammasome
AUTHOR: Lamkanfi Mohamed; Mueller James L; Vitari Alberto C; Misaghi
  Shahram; Fedorova Anna; Deshayes Kurt; Lee Wyne P; Hoffman Hal M; Dixit
  Vishva M (Reprint)
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JOURNAL: Journal of Cell Biology  187 (1): p61-70 OCT 5 2009 2009
ITEM IDENTIFIER: doi:10.1083/jcb.200903124
ISSN: 0021-9525
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Inflammasomes activate caspase-1 for processing and secretion of
  the cytokines interleukin-1 beta (IL-1 beta) and   ***IL***  -  ***18***  .
  Cryopyrin/NALP3/NLRP3 is an essential component of inflammasomes
  triggered by microbial ligands, danger-associated molecular patterns
  (DAMPs), and crystals. Inappropriate Cryopyrin activity has been
  incriminated in the pathogenesis of gouty arthritis, Alzheimer's,
  and silicosis. Therefore, inhibitors of the Nalp3 inflammasome offer
  considerable therapeutic promise. In this study, we show that the type 2
  diabetes drug glyburide prevented activation of the Cryopyrin
  inflammasome. Glyburide's cyclohexylurea group, which binds to adenosine
  triphosphatase (ATP)-sensitive K+ (K-ATP) channels for insulin secretion,
  is dispensable for inflammasome inhibition. Macrophages lacking K-ATP
  subunits or ATP-binding cassette transporters also activate the Cryopyrin
  inflammasome normally. Glyburide analogues inhibit ATP- but not
  hypothermia-induced IL-1 beta secretion from human monocytes expressing
  familial cold-associated autoinflammatory syndrome associated Cryopyrin
  mutations, thus suggesting that inhibition occurs upstream of Cryopyrin.
  Concurrent with the role of Cryopyrin in endotoxemia, glyburide
  significantly delays lipopolysaccharide-induced lethality in mice.
  Therefore, glyburide is the first identified compound to prevent
  Cryopyrin activation and microbial ligand-, DAMP-, and crystal-induced
  IL-1 beta secretion.


 7/7/2     (Item 2 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
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0021193754   BIOSIS NO.: 200900535191
EFFECTS OF INTERLEUKIN-18 ON HUMAN SH-SY5Y NEUROBLASTOMA CELL LINE
AUTHOR: Sutinen E M (Reprint); Ojala J O; Korolainen M; Pirttila T
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Finland**Finland
JOURNAL: Journal of Neurochemistry 110 (Suppl. 2): p203 SEP 2009 2009
CONFERENCE/MEETING: 22nd Biennial Meeting of the
International-Society-of-Neurochemistry/Asian-Pacific-Society-for-Neurochem
istry Busan, SOUTH KOREA August 23 -29, 2009; 20090823
SPONSOR: Int Soc Neurochem
Asian Pacific Soc
ISSN: 0022-3042
DOCUMENT TYPE: Meeting; Meeting Poster
RECORD TYPE: Citation
LANGUAGE: English

7/7/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0020855691 BIOSIS NO.: 200900196025
Interleukin-18 promoter polymorphisms and risk of late onset
Alzheimer's disease
AUTHOR: Yu Jin-Tai; Tan Lan (Reprint); Song Jing-Hui; Sun Yan-Ping; Chen
Wei; Miao Dan; Tian Yan
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Municipal Hosp, 5 Donghai Middle Rd, Qingdao 266071, Shandong, Peoples R
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AUTHOR E-MAIL ADDRESS: dr.tanlan@163.com
JOURNAL: Brain Research 1253 p169-175 FEB 9 2009 2009
ITEM IDENTIFIER: doi:10.1016/j.brainres.2008.11.083
ISSN: 0006-8993
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Pro- and anti-inflammatory cytokines play an important role in
Alzheimer's disease (AD), and common polymorphisms of genes
controlling their production have been shown to be associated with the
susceptibility to sporadic AD. Interleukin (***IL***)- ***18*** is a
potent pro-inflammatory cytokine of the IL-1 superfamily, and increasing
evidences indicate a crucial role for it in the pathogenesis of AD. To
clarify the role of IL-18 as a potential cause for AD
susceptibility, we investigated the effect of two functional
polymorphisms in IL-18 promoter: -607 C/A (rs1946518) and
-137 G/C (rs187238) for the risk of sporadic late onset Alzheimer's
disease (LOAD) in a Han Chinese population of 109 patients and 109
healthy controls matched for sex and age. All 218 subjects were also
genotyped for the Apolipoprotein E (ApoE) polymorphisms. The results
revealed that both -607 C allele and -137 G allele were associated with
an increased risk of LOAD (odds ratios/OR=1.56, P=0.04, Power=0.96 and
OR=1.85, P=0.03, Power=0.80, respectively), and these associations were
influenced by the presence of ApoE epsilon 4 alleles. Moreover, they
showed a highly significant synergistical interaction with the ApoE
epsilon 4 allele (OR=5.70 and 4.64, respectively). Examination of the
haplotypes identified the -607 C/-137 G haplotype to increase the risk of
LOAD (OR=1.62, P=0.003, Power=0.97). These findings suggest that the
functional polymorphisms in IL-18 promoter may be involved in
the risk of developing sporadic LOAD in the Han Chinese population. (C)
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7/7/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0020778926 BIOSIS NO.: 200900119260
Amyloid beta peptide promotes differentiation of pro-inflammatory human myeloid dendritic cells
AUTHOR: Ciaramella Antonio; Sanarico Nunzia; Bizzoni Federica; Moro Maria Luisa; Salani Francesca; Scapighati Giuseppe; Spalletta Gianfranco; Caltagirone Carlo; Bossu Paola (Reprint)
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AUTHOR E-MAIL ADDRESS: a.ciaramella@hsantalucia.it; sanarico@science.uniroma2.it; f.bizzoni@hsantalucia.it; ml.moro@hsantalucia.it; f.salani@hsantalucia.it; scapigg@unitus.it; g.spalletta@hsantalucia.it; c.caltagirone@hsantalucia.it; p.bossu@hsantalucia.it
JOURNAL: Neurobiology of Aging 30 (2): p210-221 FEB 2009 2009
ITEM IDENTIFIER: doi:10.1016/j.neurobiolaging.2007.06.007
ISSN: 0197-4580
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: A key event of Alzheimer's disease (AD) pathogenesis is the production of amyloid beta peptides (A beta), which are hypothesized to lead to neurodegeneration by still unclear mechanisms, including a chronic inflammatory response characterized by innate immune cell activation and pro-inflammatory molecule release. Since dendritic cells (DCs) at are central players of innate immune response and brain dendritic-like cells may have a crucial role in AD Pathogenesis, this study investigates the effects of A beta on human DC functions. Myeloid DCs differentiated in the presence of A beta 42 showed an increase in survival and soluble antigen uptake, a reduction in HLA molecule expression and in IL-10 and IL-12 production. Accordingly, A beta 42-trcated DCs were impaired in inducing T cell proliferation and IL-2 production. On the other hand, A beta 42 treatment provided DCs with the ability to release higher levels of IL-1 beta, IL-6 and IL-18 , than control DCs. These results demonstrate that A beta 42 call modulate the immune system by inducing pro-inflammatory DC differentiation, thus gaining new insights into AD pathogenesis and immune-based therapeutic intervention. (C) 2007 Elsevier Inc. All rights reserved.

7/7/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0020778925 BIOSIS NO.: 200900119259
Expression of interleukin-18 is increased in the brains of Alzheimer 's disease patients
AUTHOR: Ojala Johanna (Reprint); Alafuzoff Irina; Herukka Sanna-Kaisa; van Groen Thomas; Tanila Heikki; Pirttila Tuula
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AUTHOR E-MAIL ADDRESS: Johanna.Ojala@uku.fi; Irina.Alafuzoff@uku.fi; Sanna-Kaisa.Herukka@uku.fi; vangroen@uab.edu; Heikki.Tanila@uku.fi; Tulla.Pirttila@uku.fi
JOURNAL: Neurobiology of Aging 30 (2): p198-209 FEB 2009 2009
ITEM IDENTIFIER: doi:10.1016/j.neurobiolaging.2007.06.006
ISSN: 0197-4580
DOCUMENT TYPE: Article

RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The inflammatory cytokines can initiate nerve cell degeneration and enhance the plaque production typically found in Alzheimer's disease (AD). Interleukin-19 (***IL*** - ***18***) is an inflammatory cytokine, which call induce the expression of interferon-gamma. This interleukin shares similarities with the IL-1 family of proteins. Like IL-1 beta, IL-18 is cleaved by caspase-1 (ICE) to all active secreted form. We examined the expressions of ***IL*** - ***18*** , -1 beta and ICE in different brain regions from AD patients that were categorized with respect to the Braak stage, and age-matched with non-demented controls. The levels of total-RNA and protein of ***IL*** - ***18*** and ICE were increased, especially in the frontal lobe of AD patients and this change was not modified by ApoE genotype. Immunohistochemistry of AD brain samples detected IL-18 in microglia, astrocytes, and surprisingly in neurons, and it is also co-localized not only with amyloid-P plaques but also with tau. In CSF, elevated ***IL*** - ***18*** level was detected only in men and it also correlated with CSF tau ill MCI. ***IL*** - ***18*** may thus be a potential biomarker for men. Plasma levels of ***IL*** - ***18*** showed no correlation with the disease. In Conclusion, amyloid-P may induce the synthesis of IL-18, and IL-18 kinases involved in tau phosphorylation as a part of the amyloid-associated inflammatory reaction. (C) 2007 Elsevier Inc. All rights reserved.

7/7/6 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0020776679 BIOSIS NO.: 200900117013
Interleukin-18 increases expression of kinases involved in tau phosphorylation in SH-SY5Y neuroblastoma cells
AUTHOR: Ojala Johanna O (Reprint); Sutinen Elina M; Salminen Antero; Pirttila Tuula
AUTHOR ADDRESS: Univ Kuopio, Clin Res Ctr, Brain Res Unit, POB 1627, FIN-70211 Kuopio, Finland**Finland
AUTHOR E-MAIL ADDRESS: Johanna.Ojala@uku.fi
JOURNAL: Journal of Neuroimmunology 205 (1-2): p86-93 DEC 15 2008 2008
ITEM IDENTIFIER: doi:10.1016/j.jneuroim.2008.09.012
ISSN: 0165-5728
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Inflammatory cytokines, produced mainly by activated microglia in the brain, can enhance neuronal degeneration and the amyloid-beta-plaque production involved in ***Alzheimer*** 's disease (AD). We previously demonstrated that the expression of the pro-inflammatory cytokine interleukin-18 (IL-18) colocalizes with plaques and hyperphoshorylated tau containing neurons in AD patients. Here we exposed neuron-like, differentiated SH-SY5Y neuroblastomas to IL-18 and observed that the protein levels of p35, Cdk5, GSK-3 beta, and Ser 15-phosphorylated p53 increased during 6 h-24 h. Tau phosphorylation and expression of cyclin G1, involved in neuronal regeneration. increased at 72 h. In vivo, over-expression of ***IL*** - ***18*** may induce hyperphosphorylation of tau and induce cell cycle activators. (C) 2008 Elsevier B.V. All rights reserved.

7/7/7 (Item 7 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0020701946 BIOSIS NO.: 200900042280
Amyloid-beta oligomers set fire to inflammasomes and induce Alzheimer 's pathology
AUTHOR: Salminen Antero (Reprint); Ojala Johanna; Suuronen Tiina; Kaarniranta Kai; Kauppinen Anu
AUTHOR ADDRESS: Univ Kuopio, Dept Neurol, Inst Clin Med, POB 1627, FIN-70211 Kuopio, Finland**Finland
AUTHOR E-MAIL ADDRESS: antero.salminen@uku.fi
JOURNAL: Journal of Cellular and Molecular Medicine 12 (6A): p2255-2262 DEC 2008 2008
ITEM IDENTIFIER: doi:10.1111/j.1582-4934.2008.00496.x
ISSN: 1582-1838
DOCUMENT TYPE: Article; Literature Review
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Genetic and molecular studies have confirmed the central role of amyloid-beta production and fibrillation in the pathogenesis of ***Alzheimer*** 's disease (AD). However, the pathological pathways from amyloid-beta peptide oligomerization to the major pathological hallmarks of AD, such as neurofibrillary tangles, inflammation and loss of cholinergic neurons, are largely unknown. The innate immunity defence system utilizes pattern recognition receptors to respond to a variety of danger- and pathogen-associated molecular structures. Amyloid-beta oligomers and fibrils and their cellular effects can activate the innate immunity defence and induce inflammatory and apoptotic responses in human brain. Amyloid-beta oligomers can interfere with many aspects of neuronal membrane functions and can evoke potassium (K+) efflux from neurons. A low K+ concentration is a potent activator for the NALP1 inflammasomes, which then stimulate caspase-1 to cleave the proforms of IL-1 beta and ***IL*** - ***18*** cytokines. Interestingly, recent observations have demonstrated that amyloid-beta fibrils can activate NALP3 inflammasomes Via the lysosomal damage in mouse microglia. We will review here the activation mechanisms of NALP inflammasomes in neurons and microglia and several downstream effects in brain demonstrating that toxic amyloid-beta oligomers and fibrils can light afire in inflammasomes and induce ***Alzheimer*** 's pathology.

7/7/8 (Item 8 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0020364040 BIOSIS NO.: 200800410979
Interleukin-18 produced by peripheral blood cells is increased in Alzheimer's disease and correlates with cognitive impairment
AUTHOR: Bossu Paola (Reprint); Ciaramella Antonio; Salani Francesca; Bizzoni Federica; Varsi Erika; Di Julio Fulvia; Giubilei Franco; Gianni Walter; Trequattrini Alberto; Moro Maria Luisa; Bernardini Sergio; Caltagirone Carlo; Spalletta Gianfranco
AUTHOR ADDRESS: IRCCS, Santa Lucia Fdn, Dept Clin and Behav Neurol, Via Ardeatina 306, I-00179 Rome, Italy**Italy
AUTHOR E-MAIL ADDRESS: p.bossu@hsantalucia.it
JOURNAL: Brain Behavior and Immunity 22 (4): p487-492 MAY 2008 2008
ITEM IDENTIFIER: doi:10.1016/j.bbi.2007.10.001
ISSN: 0889-1591
DOCUMENT TYPE: Article

RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: A body of evidence indicates that inflammation plays a pivotal role in AD pathogenesis. ***IL*** - ***18*** is a pro-inflammatory cytokine produced in the brain, emerging to be implicated in AD. Although no differences in circulating IL-18 levels were measured between AD patients and controls, a significant increased production of IL-18 was obtained from stimulated blood mononuclear cells of AD patients. This was true particularly in AD subjects carrying the C/C genotype at the -607 position of ***IL*** - ***18*** gene promoter. Furthermore, a significant correlation between IL-18 production and cognitive decline was observed in AD patients. Overall, these data indicate that IL-18-related inflammatory pathways, probably also in virtue of polymorphic IL-18 gene influence, are exacerbated in AD patients, and that this cytokine may indeed participate in pathogenic processes leading to dementia. (c) 2007 Elsevier Inc. All rights reserved.

7/7/9 (Item 9 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0020066544 BIOSIS NO.: 200800113483
Pro-inflammatory cytokines and their effects in the dentate gyrus
BOOK TITLE: Progress in Brain Research
AUTHOR: Pickering Mark; O'Connor John J (Reprint)
BOOK AUTHOR/EDITOR: Scharfman HE (Editor)
AUTHOR ADDRESS: Univ Coll Dublin, Conway Inst Biomol and Biomed Res, UCD Sch Biomol and Biomed Sci, Dublin 4, Ireland**Ireland
AUTHOR E-MAIL ADDRESS: john.oconnor@ucd.ie
SERIES TITLE: PROGRESS IN BRAIN RESEARCH 163 p339-354 2007
ITEM IDENTIFIER: doi:10.1016/S0079-6123(07)63020-9
BOOK PUBLISHER: ELSEVIER SCIENCE BV, SARA BURGERHARTSTRAAT 25, PO BOX 211, 1000 AE AMSTERDAM, NETHERLANDS
ISSN: 0079-6123_(print) ISBN: 978-0-444-53015-8 (H)
DOCUMENT TYPE: Book Chapter
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The older notion of a central nervous system existing in essential isolation from the immune system has changed dramatically in recent years as the body of evidence relating to the interactions between these two systems has grown. Here we address the role of a particular subset of immune modulatory molecules, the pro-inflammatory cytokines, in regulating neuronal function and viability in the dentate gyrus of the hippocampus. These inflammatory mediators are known to be elevated in many neuropathological conditions, such as Alzheimer's disease, Parkinson's disease and ischaemic injury that follows stroke. Pro-inflammatory cytokines, such as tumour necrosis factor-alpha (TNF-alpha), interleukin 1-beta (IL-1 beta) and interleukin 18 (IL-18), have been shown to regulate neurotoxicity; although, due to the complexity of the cytokine action in neurons and glia, the effect may be either facilitatory or protective, depending on the circumstances. As well as their role in neurotoxicity and neuroprotection, the pro-inflammatory cytokines have also been shown to be potent regulators of synaptic function. In particular, TNF-alpha, IL-1 beta and ***IL*** - 18 have all been shown to inhibit long-term potentiation, a form of neuronal plasticity widely believed to underlie learning and memory, both in the early p38 mitogen activated protein kinase-dependant phase and the

later protein synthesis-dependant phase. In this article we address the mechanisms underlying these cytokine effects in the dentate gyrus of the hippocampus.

7/7/10 (Item 10 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0019991585 BIOSIS NO.: 200800038524
Altered plasma cytokine levels in Alzheimer's disease: Correlation with the disease progression
AUTHOR: Motta M; Imbesi R; Di Rosa M; Stivala F; Malaguarnera L (Reprint)
AUTHOR ADDRESS: Dept Biomed Sci, Via E De Amicis 24, I-95039 Trecastagni Catania, Italy**Italy
AUTHOR E-MAIL ADDRESS: lucmal@mbox.unict.it
JOURNAL: Immunology Letters 114 (1): p46-51 NOV 30 2007 2007
ITEM IDENTIFIER: doi:10.1016/j.imlet.2007.09.002
ISSN: 0165-2478
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Increasing evidence supports a propensity towards inflammation in ***Alzheimer*** 's disease (AD) pathogenesis. In our previous studies we observed high levels of IL-16, IL-18 and TGF-beta 1 mRNA expression in monocyte-macrophages of the peripheral blood of AD patients. The aim of this investigation was to determine the plasma levels of IL-12, IL-16, IL-18 and TGF-beta 1 in AD patients at different stages of the disease and to correlate the production of these cytokines with the disease progression. The levels of IL-12, IL-16, IL-18 and TGF-beta 1 resulted higher in AD-mild patients, were slightly lower in AD-moderate patients, whereas no significant difference was observed between AD-severe patients and non-demented age-matched subjects. The correlation values between cytokine plasma levels were dependent on the disease progression. Our data indicate that plasma levels of these inflammatory molecules follow the degree of AD suggesting a gradual decline of immune responsiveness in AD. (c) 2007 Elsevier B.V. All rights reserved.

7/7/11 (Item 11 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0019836473 BIOSIS NO.: 200700496214
Interleukin 18 gene polymorphisms predict risk and outcome of Alzheimer's disease
AUTHOR: Bossu Paola (Reprint); Ciaramella Antonio; Moro Maria Luisa; Bellincampi Lorenza; Bernardini Sergio; Federici Giorgio; Trequattrini Alberto; Macciardi Fabio; Spoletini Ilaria; Di Iulio Fulvia; Caltagirone Carlo; Spalletta Gianfranco
AUTHOR ADDRESS: IRCCS, Santa Lucia Fdn, Dept Clin and Behav Neurol, Expt Neuropsychobiol Lab, Via Ardeatina 306, I-00179 Rome, Italy**Italy
AUTHOR E-MAIL ADDRESS: p.bossu@hsantalucia.it
JOURNAL: Journal of Neurology Neurosurgery & Psychiatry 78 (8): p807-811 AUG 2007 2007
ITEM IDENTIFIER: doi:10.1136/jnnp.2006.103242
ISSN: 0022-3050
DOCUMENT TYPE: Article
RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: Background and aim: Inflammation has been extensively implicated in the pathogenesis of ***Alzheimer*** 's disease (AD). Although there is evidence of a key role for cytokines in neuroinflammation processes, so far the proinflammatory cytokine interleukin (IL)-18 has not been associated with AD. The aim of this study was to investigate the impact of two polymorphisms of the human IL-18 gene promoter at positions 2607 (C/A) and 2137 (G/C) on both susceptibility to and progression of AD.Results: The results revealed that the genotype distribution of the -607 (C/A) polymorphism was different between patients with AD and control subjects (chi(2) = 7.99, df = 2, p = 0.0184). In particular, carriers of the CC genotype were at increased risk of developing AD (OR 2.33; 95% CI 1.29 to 4.22; p = 0.0052). The observed genotypes were in Hardy-Weinberg equilibrium, as for the 2607 polymorphism, whereas the 2137 polymorphism appeared in Hardy-Weinberg disequilibrium only in the patient group (p = 0.0061). Finally, in a 2 year follow-up study, the 2137 CC genotype was strongly and specifically associated with a faster cognitive decline (F = 4.024; df = 4,192; p = 0.0037 for time by ***IL*** - ***18*** -137 G/C group interaction) with no interaction effect with the apolipoprotein E epsilon 4/non-epsilon 4 allele presence.Conclusion: As ***IL*** - ***18*** cytokine promoter gene polymorphisms have been previously described to have functional consequences on IL-18 expression, it is possible that individuals with a prevalent IL-18 gene variant have a dysregulated immune response, suggesting that IL-18 mediated immune mechanisms may play a crucial role in AD.

7/7/12 (Item 12 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

19144546 BIOSIS NO.: 200600489941
16th Nippon-Medical-School-Foundation Academic Meeting for Foreign Researchers, Tokyo, JAPAN
AUTHOR: Anonymous
JOURNAL: Journal of Nippon Medical School 73 (2): p106-111 APR 2006 2006
CONFERENCE/MEETING: 16th Nippon-Medical-School-Foundation Academic Meeting for Foreign Researchers Tokyo, JAPAN 20051119,
SPONSOR: Nippon Med Sch Fdn
ISSN: 1345-4676
DOCUMENT TYPE: Meeting; Meeting Summary
RECORD TYPE: Citation
LANGUAGE: English

7/7/13 (Item 13 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

19121861 BIOSIS NO.: 200600467256
Chitotriosidase and inflammatory mediator levels in Alzheimer's disease and cerebrovascular dementia
AUTHOR: Di Rosa Michelino; Dell'Ombra Nicola; Zambito Anna Maria; Malaguarnera Mariano; Nicoletti Ferdinando; Malaguamera Lucia (Reprint)
AUTHOR ADDRESS: Via E De Amicis 24, I-95039 Catania, Italy**Italy
AUTHOR E-MAIL ADDRESS: lucmal@mbox.unict.it
JOURNAL: European Journal of Neuroscience 23 (10): p2648-2656 MAY 2006 2006
ISSN: 0953-816X

DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Inflammation has been reported to be involved in the pathogenesis of cerebrovascular dementias (CvDs). This study investigated the involvement of Chitotriosidase (ChT), a chinolitic enzyme mainly produced by activated macrophages, in the pathophysiology of Alzheimer's disease (AD) and ischemic CvD. In addition, the levels of interleukin (IL)-16, IL-18, transforming growth factor (TGF) -beta 1 and superoxide anion (O-2(-)) were determined to evaluate the relationship between ChT levels, these cytokines and oxidative stress in both AD and ischemic CvD patients. The levels of ChT and IL-16, ***IL*** - ***18*** , and TGF-beta 1 mRNA were investigated using quantitative real-time polymerase chain reaction on macrophages of peripheral blood of 40 patients with AD, 40 patients with ischemic CvD and 40 non-demented age-matched subjects. The results show that ChT, IL-16 and O-2(-) levels significantly increased in ischemic CvD patients compared with AD patients and were significantly and positively correlated with IL- ***18*** and O-2(-). The production of ***IL*** - ***18*** was increased in both AD and ischemic CvD patients. TGF-beta 1 expression was higher in AD patients and was inversely correlated with the expression of ChT, IL-16 and ***IL*** - ***18*** , respectively. In non-demented age-matched subjects no significant changes in ChT and IL-16, IL-18, and TGF-beta 1 expression were found. Our results indicate that ChT, IL-16, ***IL*** - 18 and TGF-beta 1 are increased in ischemic CvD and AD, confirming that the immune system may play an important role in the development and progression of neurodegenerative disorders. In addition, the present findings suggest that ChT could also play a crucial role in pathological conditions such as CvD in which the inflammatory process is activated.

7/7/14 (Item 14 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18774921 BIOSIS NO.: 200600120316
High cholesterol diet results in increased expression of interleukin-6 and caspase-1 in the brain of apolipoprotein E knockout and wild type mice
AUTHOR: Rahman S M A; Van Dam A-M; Schultzberg M; Crisby M (Reprint)
AUTHOR ADDRESS: Karolinska Univ Hosp Huddinge, Karolinska Inst, Div Expt Geriatr, Neurotec Dept, SE-14186 Stockholm, Sweden**Sweden
AUTHOR E-MAIL ADDRESS: Milita.Crisby@neurotec.ki.se
JOURNAL: Journal of Neuroimmunology 169 (1-2): p59-67 DEC 2005 2005
ISSN: 0165-5728
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Inflammation in the central nervous System is an early hallmark of many neurodegenerative diseases including Alzheimer's disease (AD). Recently, increasing evidence Suggests that hypercholesterolemia during midlife and abnormalities in the cholesterol metabolism could have an important role ill the pathogenesis of AD. In the present Study, we have evaluated the effect of high cholesterol (HQ diet on the expression of interleukin-6 (IL-6), a cytokine involved in neurodegeneration, and caspase-1, that is responsible for the cleavage of the precursors of interleukin-1 beta (IL-1 beta) and interleukin-18 (IL-18) in the brain of apolipoprotein E (Apo F) knock-out (KO) and wild type (WT) mice.The density of IL-6-positive cells was increased in the hippocampus

(p<0.0001) and the dorsal part of the cortex (p<0.001) of KO and WT mice oil HC diet (KOHC and WTHC mice, respectively) compared to KO and WT mice oil ND (KOND and WTND mice, respectively). KOHC mice had increased caspase-1 positive cells and staining intensity in the hippocampus in comparison with WTHC mice (p<0.01). In the hippocampus, the density of caspase-1 positive cells was also higher in KOHC compared to KOND mice (p<0.05) and KOHC compared with WTHC mice (p<0.01). There was a major increase in caspase-1 immunoreactivity and cell density ill both the dosal part of the cortex (p<0.001) and the lateral part of the cortex (p<0.005) in KO and WT mice on HC diet compared to ND.The findings of the present study indicate that Chronic exposure to HC diet increases the expression of the two important inflammatory mediators IL-6 and caspase-1 in the brain of KO and WT mice. In the case of caspase-1, we report a major difference in the effect of HC diet on the KO mice compared to WT mice in the hippocampus. Increased expression of inflammatory mediators involved in neurodegeneration could be a potential mechanism by which hypercholesterolemia and HC diet increase the risk of AD. (C) 2005 Elsevier B.V. All rights reserved.

7/7/15 (Item 15 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18387987 BIOSIS NO.: 200510082487
Soluble interleukin-1 receptor type II, IL-18 and caspase-1 in mild cognitive impairment and severe Alzheimer's disease
AUTHOR: Lindberg Catharina; Chromek Milan; Ahrengart Leif; Brauner Annelie; Schultzberg Marianne; Garlind Anita (Reprint)
AUTHOR ADDRESS: Karolinska Univ Hosp, Karolinska Inst, Div Clin Geriatr, Dept Neurotec, SE-14186 Stockholm, Sweden**Sweden
AUTHOR E-MAIL ADDRESS: Anita.Garlind@neurotec.ki.se
JOURNAL: Neurochemistry International 46 (7): p551-557 JUN 05 2005
ISSN: 0197-0186
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: In the present study, we have determined levels of soluble interleukin-1 (IL-1) receptor type II (sIL-1RII), interleukin-18 (IL-18) and caspase-1 in cerebrospinal fluid and serum from mild cognitive impairment patients that later progressed to ***Alzheimer*** 's disease (AD) and severe AD patients. Previous studies have shown that a chronic local inflammatory process is a part of AD neuropathology. In this process, activated microglial production of IL-1 seems to play an important role. In a previous study, we have shown increased levels of sIL-1RII in CSF from AD patients in a mild-moderate disease stage. In the present study, we found no significant differences in CSF or serum levels of sIL-1RII in either mild cognitive impairment or advanced AD patients as compared to control subjects. Likewise, there was no significant difference between mild cognitive impairment and severe AD patients. The same was true for caspase-1 and ***IL*** - ***18*** serum levels, whereas CSF levels of caspase-1 and IL-18 were below detection limits. Our data indicate that the IL-1 system is relatively intact in the early and late stages of AD. (c) 2005 Elsevier Ltd. All rights reserved.

7/7/16 (Item 16 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17762932 BIOSIS NO.: 200400143689
P2X7 receptor modulation of beta-amyloid- and LPS-induced cytokine secretion from human macrophages and microglia.
AUTHOR: Rampe David; Wang Lin; Ringheim Garth E (Reprint)
AUTHOR ADDRESS: Department of Immunology, Aventis Pharmaceuticals, Inc., Route 202-206, Mail Stop: G303A, Bridgewater, NJ, 08807-0800, USA**USA
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JOURNAL: Journal of Neuroimmunology 147 (1-2): p56-61 February 2004 2004
MEDIUM: print
ISSN: 0165-5728 _(ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: To test whether extracellular ATP can play a role in the neuroimmunopathology of Alzheimer's disease (AD), we evaluated the capacity of the ATP-binding purinoreceptor, P2X7, to modulate cytokine secretion on cultured human macrophages and microglia pre-activated 24 h with the 42 amino acid beta-amyloid peptide (Abeta(1-42)) or lipopolysaccharide. Thirty minutes of exposure to the selective P2X7 agonist 2'-3'-O-(4-benzoylbenzoyl)adenosine 5'-triphosphate (BzATP) resulted in the secretion of IL-1beta after either Abeta(1-42) or LPS stimulation of human macrophages that was dependent on the concentration of the stimulus used to pre-activate the cells. Further tests on human microglia treated with BzATP (300 muM) resulted in a 1.5- and 3.5-fold enhancement of IL-1alpha and IL-1beta secretion, respectively, from cells pre-activated by 10 muM Abeta(1-42) and a 1.6- and 3.9-fold enhancement of IL-1alpha and IL-1beta secretion, respectively, from cells pre-activated by 1 mug/ml LPS. BzATP induction of IL-1alpha and IL-1beta secretion from microglia was completely reversed by pre-incubation of the cells with the P2X7 antagonist, adenosine 5-triphosphate 2',3'-acyclic dialcohol (oxidized ATP). In contrast to its effects on IL-1alpha and IL-1beta secretion, BzATP induced TNF-alpha after LPS stimulation, but not after stimulation with Abeta(1-42), induced IL-18 secretion regardless of whether microglia were pre-activated and attenuated IL-6 secretion after either LPS or Abeta(1-42) pre-activation. These results demonstrate that extracellular ATP can modulate Abeta-induced cytokine secretion from human rnacrophages and microglia and thus may play a role in the neuroimmunopathology of AD.

7/7/17 (Item 17 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17516158 BIOSIS NO.: 200300484877
Role of cytokines in neurological disorders.
AUTHOR: Aarli Johan A (Reprint)
AUTHOR ADDRESS: Department of Neurology, University of Bergen, Bergen, Norway**Norway
AUTHOR E-MAIL ADDRESS: johan.a.aarli@helse-bergen.no
JOURNAL: Current Medicinal Chemistry 10 (19): p1931-1937 October 2003 2003
MEDIUM: print
ISSN: 0929-8673
DOCUMENT TYPE: Article; Editorial
RECORD TYPE: Citation
LANGUAGE: English

7/7/18 (Item 18 from file: 5)

DIALOG(R)File 5:Biosis Previews(R)

17346301 BIOSIS NO.: 200300303790
HIPPOCAMPAL INJURY AND GLIAL ACTIVATION WITHOUT UP - REGULATION OF INFLAMMATORY MEDIATORS: A GENE ARRAY ANALYSIS.
AUTHOR: Little A R (Reprint); O'Callaghan J P (Reprint)
AUTHOR ADDRESS: CDC-NIOSH, Morgantown, WV, USA**USA
JOURNAL: Society for Neuroscience Abstract Viewer and Itinerary Planner 2002 pAbstract No. 415.1 2002 2002
MEDIUM: cd-rom
CONFERENCE/MEETING: 32nd Annual Meeting of the Society for Neuroscience Orlando, Florida, USA November 02-07, 2002; 20021102
SPONSOR: Society for Neuroscience
DOCUMENT TYPE: Meeting; Meeting Poster; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Damage to the CNS results in a complex series of molecular and cellular events involving the injured cells and their supportive environment. Often, the response observed is complicated by factors extrinsic to CNS, such as an influx of blood-borne elements. We have used systemic administration of trimethyltin (TMT) (8.0 mg/kg) to damage the rat hippocampus without disrupting the blood brain barrier. This injury model causes extensive loss of hippocampal neurons and an accompanying microglial and astroglial activation, effects often linked to inflammatory responses (e.g. in ***Alzheimers*** disease). Previously, we have shown that TMT causes a marked and early up-regulation of the chemokine, MCP-1, in microglia. Surprisingly, expression of MCP-1 appeared to occur without an induction of other chemokines or proinflammatory cytokines. Thus, we now have used the Affymetrix gene chip as a more comprehensive approach for analysis of early gene expression patterns in the TMT injury model. At 2 and 5 days post TMT our findings confirm the induction of MCP-1 as well as a number of microglial (MHCII Ia antigen) and astroglial (GFAP, peripheral benzodiazepine receptors, glutamine synthetase, vimentin) genes. Genes implicated in the regulation of MCP-1 expression (p38 and JNK2 kinases), however, were not affected, whereas genes implicated in down stream effects of MCP-1 (ERK1 and PI3Kinase) were increased. Notably, genes associated with proinflammatory responses (e.g. TNF, IL-1, -2, -7, -9, -10, -12, -13, -15, -18, IFN, and complement) were not affected. These findings implicate a role for MCP-1 in hippocampal injury without the participation of other proinflammatory chemokines and cytokines.

7/7/19 (Item 19 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16429347 BIOSIS NO.: 200200022858
Modulation of cytokine release and pore formation by P2X7 channels in beta amyloid-activated human macrophages
AUTHOR: Ringheim G E (Reprint); Wang L; Rampe D
AUTHOR ADDRESS: Immunology, Aventis Pharmaceuticals, Inc, Bridgewater, NJ, USA**USA
JOURNAL: Society for Neuroscience Abstracts 27 (2): p2558 2001 2001
MEDIUM: print
CONFERENCE/MEETING: 31st Annual Meeting of the Society for Neuroscience San Diego, California, USA November 10-15, 2001; 20011110
ISSN: 0190-5295
DOCUMENT TYPE: Meeting; Meeting Abstract

RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The P2Z/P2X7 receptor is a subtype of purinoreceptor expressed on the surface of macrophages and microglia, and which functions as an ATP ligand-gated ion channel. Expression of P2X7 can be upregulated by lipopolysaccharide (LPS), which in the presence of ATP, can enhance release of the cytokines IL-1beta and ***IL*** - ***18*** . We investigated the effects of the P2X7 agonist 3'-O-(4-benzoylbenzoyl)-adenosine 5'-triphosphate (BzATP) on human peripheral blood-derived macrophages that had been activated for 5 h by the beta amyloid (1-42) peptide (10 muM) implicated in the pathology of Alzheimer's disease, and we compared this to the effects of LPS. Exposure of these cells to BzATP lead to a concentration-dependent (1-1000 muM) rapid release at 30 min of IL-1alpha IL-1beta, and ***IL*** - ***18*** . At 300 muM BzATP, respective increases were IL-1alpha (>five-fold), IL-1beta (>five-fold) and IL - ***18*** (>two-fold), but none for IL-6 or TNF-alpha. Cytokine release was blocked >80% by 300 muM oxidized adenosine triphosphate (oxATP), a P2X7 antagonist. Treatment of the cells with oxATP 1 h before LPS or beta amyloid stimulation attenuated not only the extracellullar release of cytokines, but also their intracellular accumulation over 24 h, suggesting an involvement of P2X7 receptors in cytokine gene expression as well as release. We propose that the P2X7 channels on activated macrophages and, by extension, microglia surrounding deposits of beta amyloid in the brains of Alzheimer's disease patients, are participating in the inflammatory processes leading to enhanced cytokine production.

7/7/20 (Item 20 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15919731 BIOSIS NO.: 200100091570
Beneficial effect(s) of n-3 fatty acids in cardiovascular diseases: But, why and how?
AUTHOR: Das U N (Reprint)
AUTHOR ADDRESS: EFA Sciences LLC, 1420 Providence Highway, Suite No. 266, Norwood, MA, 02062, USA**USA
JOURNAL: Prostaglandins Leukotrienes and Essential Fatty Acids 63 (6): p 351-362 December, 2000 2000
MEDIUM: print
ISSN: 0952-3278
DOCUMENT TYPE: Article; Literature Review
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Low rates of coronary heart disease was found in Greenland Eskimos and Japanese who are exposed to a diet rich in fish oil. Suggested mechanisms for this cardio-protective effect focused on the effects of n-3 fatty acids on eicosanoid metabolism, inflammation, beta oxidation, endothelial dysfunction, cytokine growth factors, and gene expression of adhesion molecules; But, none of these mechanisms could adequately explain the beneficial actions of n-3 fatty acids. One attractive suggestion is a direct cardiac effect of n-3 fatty acids on arrhythmogenesis. N-3 fatty acids can modify Na+ channels by directly binding to the channel proteins and thus, prevent ischemia-induced ventricular fibrillation and sudden cardiac death. Though this is an attractive explanation, there could be other actions as well. N-3 fatty acids can inhibit the synthesis and release of pro-inflammatory cytokines such as tumor necrosis factoralpha (TNFalpha) and interleukin-1 (IL-1)

and IL-2 that are released during the early course of ischemic heart disease. These cytokines decrease myocardial contractility and induce myocardial damage, enhance the production of free radicals, which can also suppress myocardial function. Further, n-3 fatty acids can increase parasympathetic tone leading to an increase in heart rate variability and thus, protect the myocardium against ventricular arrhythmias. Increased parasympathetic tone and acetylcholine, the principle vagal neurotransmitter, significantly attenuate the release of TNF, IL-1beta, IL-6 and ***IL*** - ***18*** . Exercise enhances parasympathetic tone, and the production of anti-inflammatory cytokine IL-10 which may explain the beneficial action of exercise in the prevention of cardiovascular diseases and diabetes mellitus. TNFalpha has neurotoxic actions, where as n-3 fatty acids are potent neuroprotectors and brain is rich in these fatty acids. Based on this, it is suggested that the principle mechanism of cardioprotective and neuroprotective action (s) of n-3 fatty acids can be due to the suppression of TNFalpha and IL synthesis and release, modulation of hypothalamic-pituitary-adrenal anti-inflammatory responses, and an increase in acetylcholine release, the vagal neurotransmitter. Thus, there appears to be a close interaction between the central nervous system, endocrine organs, cytokines, exercise, and dietary n-3 fatty acids. This may explain why these fatty acids could be of benefit in the management of conditions such as septicemia and septic shock, Alzheimer's disease, Parkinson's disease, inflammatory bowel diseases, diabetes mellitus, essential hypertension and atherosclerosis.

7/7/21 (Item 21 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15587844 BIOSIS NO.: 200000306157
Induction of cytokines in glial cells surrounding cortical beta-amyloid plaques in transgenic Tg2576 mice with Alzheimer pathology
AUTHOR: Mehlhorn Gaby; Hollborn Margrit; Schliebs Reinhard (Reprint)
AUTHOR ADDRESS: Department of Neurochemistry, Paul Flechsig Institute for Brain Research, University of Leipzig, Jahnallee 59, D-04109, Leipzig, Germany**Germany
JOURNAL: International Journal of Developmental Neuroscience 18 (4-5): p 423-431 July-August, 2000 2000
MEDIUM: print
ISSN: 0736-5748
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: beta-Amyloid plaque deposition observed in brains from Alzheimer patients, might function as immune stimulus for glial/macrophages activation, which is supported by observations of activated microglia expressing interleukin (IL)-1beta and elevated IL-6 immunoreactivity in close proximity to amyloid plaques. To elucidate the mechanisms involved in beta-amyloid-mediated inflammation, transgenic mice (Tg2576) expressing high levels of the Swedish double mutation of human amyloid precursor protein and progressively developing typical beta-amyloid plaques in cortical brain regions including gliosis and astrocytosis, were examined for the expression pattern of a number of cytokines. Using ribonuclease protection assay, interleukin (IL)-1alpha,-beta, IL-1 receptor antagonist, IL-6, IL-10, IL-12, IL -18, interferon-gamma, and macrophage migration inhibitory factor (MIF) mRNA were not induced in a number of cortical areas of Tg2576 mice regardless of the postnatal ages studied ranging between 2 and 13 months. Using immunocytochemistry for IL-1alpha,beta, IL-6, tumor necrosis factor

(TNF)-alpha, and macrophage chemotactic protein (MCP)-1, only IL-1beta was found to be induced in reactive astrocytes surrounding beta-amyloid deposits detected in 14-month-old Tg2576 mice. Using non-radioactive in situ hybridization glial fibrillary acidic protein (GFAP) mRNA was detected to be expressed by reactive astrocytes in close proximity to beta-amyloid plaques. The local immune response detected around cortical beta-amyloid deposits in transgenic Tg2576 mouse brain is seemingly different to that observed in brains from Alzheimer patients but may represent an initial event of chronic neuroinflammation at later stages of the disease.

7/7/22 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE

0082365111 EMBASE No: 2008178850
The diagnostic role of serum inflammatory and soluble proteins on dementia subtypes: Correlation with cognitive and functional decline
Ozturk C.; Ozge A.; Yalin O.O.; Yilmaz I.A.; Delialioglu N.; Yildiz C.; Tesdelen B.; Kudiaki C.
Mersin University School of Medicine, Mersin University School of Science and Letters, Mersin, Turkey
AUTHOR EMAIL: aozge@mersin.edu.tr
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Behavioural Neurology (Behav. Neurol.) (Netherlands) December 1, 2007 , 18/4 (207-215)
CODEN: BNEUE ISSN: 0953-4180
DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 77

In the past years, the possible involvement of inflammation in the pathogenesis of dementia has been the subject of several investigations. However there are restricted data about the profile of the inflammatory and soluble proteins in well evaluated Alzheimer's disease (AD), vascular dementia (VD), mild cognitive impairment (MCI) and healthy controls. There are also no reliable data regarding the relationship between the overlapping protein levels and cognitive or functional decline. We measured levels of IL-1beta, IL-2, IL-6, IL-18, TNF-alpha, beta-Amlyloid 1-40 and alpha SUB 1- antichymotrypsin levels in plasma in groups of total 82 subjects with AD, MCI, VD and controls using enzyme-linked immunosorbent assay (ELISA) method. Our study samples showed high levels of proinflammatory cytokine levels (especially IL-18) in all patient groups but only high levels of alpha SUB 1- antichymotrypsine in VD patients compared to controls. There is no significant correlation between the laboratory and clinical variables except for a link between IL-1beta and NPI scores of AD. In conclusion, this study yielded evidence of some shared mechanisms underlying AD and VD and thus motivates further studies of inflammatory markers in various types of dementia and MCI. (c) 2007 - IOS Press and the authors. All rights reserved.

7/7/23 (Item 2 from file: 73)
DIALOG(R)File 73:EMBASE

0081725024 EMBASE No: 2007158831

The role of the purinergic P2X SUB 7 receptor in inflammation
Lister M.F.; Sharkey J.; Sawatzky D.A.; Hodgkiss J.P.; Davidson D.J.; Rossi A.G.; Finlayson K.
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CORRESP. AUTHOR/AFFIL: Rossi A.G.: MRC Centre for Inflammation Research, Queen's Medical Research Institute, University of Edinburgh, 47 Little France Crescent, Edinburgh, EH16 4TJ, United Kingdom
CORRESP. AUTHOR EMAIL: a.g.rossi@ed.ac.uk

Journal of Inflammation (J. Inflamm.) (United Kingdom) April 23, 2007 , 4/-
eISSN: 1476-9255
DOI: 10.1186/1476-9255-4-5
ARTICLE NUMBER: 5
DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 150

The inflammatory process, orchestrated against a variety of injurious stimuli, is composed of three inter-related phases; initiation, propagation and resolution. Understanding the interplay between these three phases and harnessing the beneficial properties of inflammation whilst preventing its damaging effects, will undoubtedly lead to the advent of much needed therapies, particularly in chronic disease states. The P2X SUB 7 receptor (P2X SUB 7R) is increasingly recognised as an important cell surface regulator of several key inflammatory molecules including IL-1beta, ***IL*** - ***18*** , TNF-alpha and IL-6. Moreover, as P2X SUB 7R-dependent cytokine production is driven by activating the inflammasome, antagonists of this receptor are likely to have therapeutic potential as novel anti-inflammatory therapies. The function of the P2X SUB 7R in inflammation, immunity and its potential role in disease will be reviewed and discussed. (c) 2007 Lister et al; licensee BioMed Central Ltd.

0081284798 EMBASE No: 2006347155
Interleukin-18 and transforming growth factor-beta 1 plasma levels in Alzheimer's disease and vascular dementia
Malaguarnera L.; Motta M.; Di Rosa M.; Anzaldi M.; Malaguarnera M.
Department of Biomedical Sciences, University of Catania, Italy; Via E. De Amicis, 24, 95039 Trecastagni-Catania, Italy
AUTHOR EMAIL: lucmal@mbox.unict.it
CORRESP. AUTHOR/AFFIL: Malaguarnera L.: Via E. De Amicis, 24, 95039 Trecastagni-Catania, Italy
CORRESP. AUTHOR EMAIL: lucmal@mbox.unict.it

Neuropathology (Neuropathology) (Australia) August 1, 2006, 26/4 (307-312)
CODEN: NOPAF ISSN: 0919-6544 eISSN: 1440-1789
DOI: 10.1111/j.1440-1789.2006.00701.x
DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 44

Inflammation has been involved in the development of dementia in cerebrovascular diseases. To investigate the cellular activation of the peripheral immune system in patients with Alzheimer's disease (AD) and vascular dementia (VaD), we determined the presence of IL-***18*** and TGF-beta1 in the plasma by using ELISA. The levels of ***IL*** -18 and TGF-beta1 were significantly elevated in patients with AD and VaD compared to non-demented, age-matched subjects. We found an inverse correlation between the levels of IL-18 and TGF-beta1 in AD patients. In VaD patients, the correlation between ***IL*** - ***18*** and TGF-beta1 reached a borderline positive value. Whereas, in the non-demented, age-matched subjects, a positive correlation between IL - ***18*** and TGF-beta1 levels was observed. These findings indicate that IL-18 and TGF-beta1 elevation is associated with AD and VaD patients, confirming that the immune system might exert a remarkable role in the development and progression of neurodegenerative disorders. Moreover, as different modifications were detected in the patients affected by AD and VaD, we propose that IL-18 and TGF- beta1 plasma levels might represent possible differential biomarkers. (c) 2006 Japanese Society of Neuropathology.

7/7/25 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

151265386 CA: 151(12)265386y PATENT
Compositions and methods for crystallizing antibody fragments
INVENTOR(AUTHOR): Argiriadi, Maria A.; Borhani, David W.; Xiang, Tao; Wu, Chengbin; Ghayur, Tariq
LOCATION: USA
ASSIGNEE: Abbott Laboratories
PATENT: PCT International ; WO 200999545 A1 DATE: 20090813
APPLICATION: WO 2009US568 (20090129) *US 2008PV62887 (20080130) *US 2008PV135739 (20080722)
PAGES: 54pp. CODEN: PIXXD2 LANGUAGE: English
PATENT CLASSIFICATIONS:
IPCR/8 + Level Value Position Status Version Action Source Office:
A61K-0039/395 A I F B 20060101 H US
DESIGNATED COUNTRIES: AE; AG; AL; AM; AO; AT; AU; AZ; BA; BB; BG; BH; BR; BW; BY; BZ; CA; CH; CN; CO; CR; CU; CZ; DE; DK; DM; DO; DZ; EC; EE; EG; ES; FI; GB; GD; GE; GH; GM; GT; HN; HR; HU; ID; IL; IN; IS; JP; KE; KG; KM; KN; KP; KR; KZ; LA; LC; LK; LR; LS; LT; LU; LY; MA; MD; ME; MG; MK; MN; MW; MX; MY; MZ; NA; NG; NI; NO; NZ; OM; PG; PH; PL; PT; RO; RS; RU; SC; SD; SE; SG; SK; SL; SM; ST; SV; SY; TJ; TM; TN; TR DESIGNATED REGIONAL: AT; BE; BG; CH ; CY; CZ; DE; DK; EE; ES; FI; FR; GB; GR; HR; HU; IE; IS; IT; LT; LU; LV; MC; MK; MT; NL; NO; PL; PT; RO; SE; SI; SK; TR; BF; BJ; CF; CG; CI; CM; GA; GN; GQ; GW; ML; MR; NE; SN; TD; TG; BW; GH; GM; KE; LS; MW; MZ; NA; SD; SL; SZ; TZ; UG; ZM; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM
SECTION:
CA215003 Immunochemistry
CA201XXX Pharmacology
CA214XXX Mammalian Pathological Biochemistry
IDENTIFIERS: crystn antibody Fab fragment interleukin 18
DESCRIPTORS:
AIDS(disease)...
-related complex; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Hypogammaglobulinemia...
acquired hypogammaglobulinemia; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of

Pain...
    acute; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Respiratory distress syndrome...
    adult; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Cirrhosis...
    alc.; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Alcoholic liver disease...
    alc. cirrhosis; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Allergy... Eye,disease... Inflammation...
    allergic conjunctivitis; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Transplant and Transplantation... Transplant rejection...
    allotransplant; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Inflammation... Spinal column,disease...
    ankylosing spondylitis; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Interleukin 18...
    antibody Fab fragment complexes; crystallization of
Erythropoiesis...
    aplasia; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Anemia...
    aplastic; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Alopecia...
    areata; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Dermatitis...
    atopic; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Allergy...
    atopy; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Autoimmune disease...
    autoimmune hemolytic anemia; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Anemia...
    autoimmune hemolytic; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Disease,animal...
    autoimmune lymphoproliferative syndrome; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Autoimmune disease... Inflammation... Thyroid gland,disease...
    autoimmune thyroiditis; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Autoimmune disease... Eye,disease... Inflammation...
    autoimmune uveitis; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Dermatitis... Hearing loss... Hypoglycemia... Hypothyroidism... Intestinal disease... Myocarditis... Thrombocytopenia... Thyroid gland,disease...
    autoimmune; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Eye,disease... Inflammation...
    blepharitis; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Bronchial disease... Obstructive pulmonary disease...

bronchiectasis; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Skin,disease...
bullous pemphigoid; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Skin,disease...
bullous; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Spinal column,disease...
cervical spondylosis; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Fatigue,biological...
chronic fatigue syndrome; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Pain... Viral hepatitis...
chronic; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Eye,disease... Inflammation...
conjunctivitis; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Addison's disease... Adrenal insufficiency... AIDS(disease)... Allergy... Alzheimer disease... Anaphylaxis... Antiphospholipid syndrome... Atherosclerosis... Autoimmune hepatitis... Autoimmune polyendocrinopathy... Behcet's syndrome... Bronchiolitis obliterans... Cachexia... Cardiovascular disease... Celiac disease... Chlamydia... Cholestasis... Chronic obstructive pulmonary disease... Connective tissue disease... Crohn disease... Demyelination... Depression... Dermatomyositis... Drug toxicity... Endocarditis... Endometriosis... Fibrosis... Glomerulonephritis... Gout... Graves' disease... Hay fever... Heart failure... Hematopoietic neoplasm... Hepatitis B... Hepatitis C... Hyperthyroidism... Hypoparathyroidism... Inflammatory bowel disease... Ischemia... Kawasaki disease... Leukemia... Leukocytopenia... Liver disease... Lyme disease... Lymphoma... Mental and behavioral disorders... Multiple organ failure... Multiple sclerosis... Myasthenia gravis... Myelodysplastic syndromes... Myocardial infarction... Myocarditis... Neoplasm... Nephritis... Nephrotic syndrome... Osteoarthritis... Ovary,neoplasm... Pancreatitis... Paralysis... Parasitic infection... Parkinson's disease... Poliomyelitis... Prostate gland,neoplasm... Prostatitis... Pulmonary fibrosis... Rheumatic fever... Rheumatic heart disease... Sarcoidosis... Schizophrenia... Scleroderma... Sepsis... Shock(circulatory collapse)... Sjogren syndrome... Stroke... Systemic lupus erythematosis... Ulcerative colitis... Urticaria... Vascular restenosis... Vitiligo... Wound healing...
crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Immune complexes...
crystallization of antibody Fab fragment complexes with interleukin-18
Human... Mouse... Mus musculus...
crystallization of antibody Fab fragments targeting interleukin-18
Disease,animal...
dacryocystitis; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Retinal disease...
diabetic retinopathy; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Cardiomyopathy...
dilated; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Cutaneous lupus erythematosus...
discoid; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Reticuloendothelial system...

disease, histiocytosis; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Spinal column,disease...
disk prolapse; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Blood coagulation disorders...
disseminated intravascular coagulation; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Eye,disease...
dry eye syndrome; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Eye,disease... Inflammation...
endophthalmitis; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Pneumonia...
eosinophilic; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Analgesics... Anti-AIDS agents...
Fab fragments of antibodies to human interleukin-18
Ovarian disease...
failure, premature, autoimmune; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Fertility disorders...
female; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Polyoxyalkylenes...
for crystallization of anti-interleukin 18 antibody Fab fragments and IL-18/Fab complexes
Protein sequences...
for heavy chain fragments and light chains of human and mouse antibodies to interleukin-18
Antibodies and Immunoglobulins...
fragments, Fab; crystallization of antibody Fab fragments targeting interleukin-18
Arteritis...
giant cell arteritis; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Kidney disease...
Goodpasture syndrome; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Transplant and Transplantation...
graft-vs.-host reaction; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Nervous system,disease...
Guillain-Barre syndrome; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Anemia...
hemolytic; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Purpura(disease)...
Henoch-Schoenlein; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Injury...
hepatic, alc.; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Disease,animal...
histiocytosis; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Disease,animal...
Hughes Syndrome; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of

Nervous system,disease...
    Huntington's chorea; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Thrombocytopenia...
    idiopathic; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Antibodies and Immunoglobulins...
    IgG1, monoclonal; crystallization of anti-interleukin 18 antibody Fab fragments and IL-18/Fab complexes of
Myositis...
    inclusion body; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Streptococcus...
    infection; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Liver disease...
    injury, alc.; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
Diabetes mellitus...
    insulin-dependent; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
  CAS REGISTRY NUMBERS:
1180118-79-9 1180118-80-2 1180118-81-3 1180118-82-4 1180118-83-5
    1180118-84-6 amino acid sequence; crystallization of anti-interleukin 18 antibody Fab fragments and IL-18/Fab complexes
64-19-7 77-92-9 288-32-4 7664-38-2 biological studies, buffer; for crystallization of anti-interleukin 18 antibody Fab fragments and IL-18/Fab complexes
7786-30-3 biological studies, for crystallization of anti-interleukin 18 antibody Fab fragments and IL-18/Fab complexes
9004-10-8 biological studies, resistance; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of
103-47-9 150-25-4 1132-61-2 1135-40-6 4432-31-9 6976-37-0 15132-04-4
    buffer; for crystallization of anti-interleukin 18 antibody Fab fragments and IL-18/Fab complexes
1179822-04-8 1179822-05-9 1179822-06-0 1179822-07-1 crystallization of anti-interleukin 18 antibody Fab fragments and IL-18/Fab complexes
77-86-1 7365-45-9 15471-17-7 25322-68-3 87993-44-0 for crystallization of anti-interleukin 18 antibody Fab fragments and IL-18/Fab complexes

 7/7/26   (Item 2 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

  143323906   CA: 143(18)323906r   CONFERENCE PROCEEDING
  CAMP-specific Pde4B mediated Aβ-induced microglial activation
  AUTHOR(S): Sebastiani, G.; Morissette, C.; Lagace, C.; Boule, M.; Ouellette, M. J.; McLaughlin, R. W.; Lacombe, D.; Gervais, F.; Tremblay, P.
  LOCATION: Neurochem (International) Limited, Laval, QC, Can., H7V 4A7
  JOURNAL: Amyloid Amyloidosis, (Int. Symp. Amyloidosis), 10th (Amyloid and Amyloidosis, (International Symposium on Amyloidosis), 10th, Tours, France, Apr. 18-22, 2004) EDITOR: Grateau, Gilles (Ed), Kyle, Robert A. (Ed), Skinner, Martha (Ed), DATE: 2005 PAGES: 382-384 CODEN: 69GPWW
  LANGUAGE: English MEETING DATE: 20040000 PUBLISHER: CRC Press LLC,Boca Raton, Fla
  SECTION:
    CA214010 Mammalian Pathological Biochemistry
  IDENTIFIERS: amyloidbeta microglia cAMP phosphodiesterase 4B gene
    Alzheimer, cDNA array technol
  DESCRIPTORS:

Neuroglia...
    microglia; soluble and fibrillary amyloid-β1-42 induced microglia cell activation upregulated cAMP-specific phosphodiesterase 4B gene expression as shown by using cDNA array technol.
Gene expression profiles,animal... DNA microarray technology...
    s- and fAβ1-42 induced microglia cell activation upregulated Pde4B, MIP-2, Lyn, Kcnk3, Scrab1, PTP1B, IL-1α, IL-18, M-ras, Nr2c2, Gsr, Slc5a2, Inpp4a, Fabp5, TNF-α, IP-10 gene express
Gene,animal... Human... Alzheimer's disease...
    soluble and fibrillary amyloid-β1-42 induced microglia cell activation up-regulated cAMP-specific phosphodiesterase 4B gene expression as shown by using cDNA array technol.
  CAS REGISTRY NUMBERS:
9036-21-9 60-92-4 107761-42-2 soluble and fibrillary amyloid-β1-42 induced microglia cell activation up-regulated cAMP-specific phosphodiesterase 4B gene expression as shown by using cDNA array technol.

 7/7/27   (Item 3 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

  142462257   CA: 142(25)462257x   PATENT
  Human antibodies to interleukin-18
  INVENTOR(AUTHOR): Ghayur, Tariq; Labkovsky, Boris; Voss, Jeffrey W.; Green, Larry; Babcook, John; Jia, Xiao-chi; Wieler, James; Kang, Jaspal Singh; Hedberg, Brad
  LOCATION: USA
  PATENT: U.S. Pat. Appl. Publ. ; US 20050100965 A1 DATE: 20050512
  APPLICATION: US 2003706689 (20031112)
  PAGES: 87 pp. CODEN: USXXCO LANGUAGE: English
  PATENT CLASSIFICATIONS:
    CLASS: 435007100; G01N-033/53A; C07H-021/04B; C07K-016/24B; C12N-005/06B
  SECTION:
    CA215001 Immunochemistry
    CA201XXX Pharmacology
    CA214XXX Mammalian Pathological Biochemistry
    CA263XXX Pharmaceuticals
  IDENTIFIERS: interleukin 18 human antibody
  DESCRIPTORS:
Skin,disease...
    acanthosis nigrans; in combination therapy with human antibodies to interleukin-18
Antibodies and Immunoglobulins...
    acquired hypogammaglobulinemia; in combination therapy with human antibodies to interleukin-18
Respiratory distress syndrome...
    adult; in combination therapy with human antibodies to interleukin-18
Cirrhosis...
    alc.; in combination therapy with human antibodies to interleukin-18
Lung,disease...
    alveolitis, cryptogenic fibrosing; in combination therapy with human antibodies to interleukin-18
Spinal column,disease... Inflammation...
    ankylosing spondylitis; in combination therapy with human antibodies to interleukin-18
Artery,disease... Inflammation...
    arteritis, giant cell; in combination therapy with human antibodies to interleukin-18

Artery,disease... Inflammation...
arteritis, Takayasu's disease; in combination therapy with human antibodies to interleukin-18
Disease,animal...
arthropathy; in combination therapy with human antibodies to interleukin-18
Allergy...
atopy; in combination therapy with human antibodies to interleukin-18
Hepatitis...
autoimmune, cryptogenic; in combination therapy with human antibodies to interleukin-18
Anemia(disease)... Autoimmune disease...
autoimmune hemolytic anemia; in combination therapy with human antibodies to interleukin-18
Autoimmune disease...
autoimmune thrombocytopenia; in combination therapy with human antibodies to interleukin-18
Thyroid gland,disease... Autoimmune disease... Inflammation...
autoimmune thyroiditis; in combination therapy with human antibodies to interleukin-18
Hypothyroidism...
autoimmune; in combination therapy with human antibodies to interleukin-18
Hepatitis...
B; in combination therapy with human antibodies to interleukin-18
Bronchi,disease... Inflammation...
bronchiolitis; in combination therapy with human antibodies to interleukin-18
Hepatitis...
C; in combination therapy with human antibodies to interleukin-18
Mycosis...
candidiasis, mucocutaneous; in combination therapy with human antibodies to interleukin-18
Antibodies and Immunoglobulins...
chimeric; to human interleukin-18
Animal cell line...
CHO; for preparation of antibodies to human interleukin-18
Biliary tract,disease...
cholestasis; in combination therapy with human antibodies to interleukin-18
Fatigue,biological...
chronic fatigue syndrome; in combination therapy with human antibodies to interleukin-18
Radionuclides,biological studies... Fluorescent substances... Luminescent substances...
conjugates with antibodies to human interleukin-18
Enzymes,biological studies...
conjugates; with antibodies to human interleukin-18
Animal cell line...
COS; for preparation of antibodies to human interleukin-18
Inflammation...
Crohn's disease; in combination therapy with human antibodies to interleukin-18
Intestine,disease...
Crohn's; in combination therapy with human antibodies to interleukin-18
Mental disorder...
depression; in combination therapy with human antibodies to interleukin-18
Peptides,biological studies...
depsipeptides, polymeric; for delivery of antibodies to human interleukin-18

Heart,disease...
    dilated cardiomyopathy; in combination therapy with human antibodies to interleukin-18
Lupus erythematosus...
    discoid; in combination therapy with human antibodies to interleukin-18
Platelet(blood)...
    disease, autoimmune thrombocytopenia; in combination therapy with human antibodies to interleukin-18
Platelet(blood)...
    disease, thrombocytopenia, idiopathic; in combination therapy with human antibodies to interleukin-18
Joint,anatomical...
    disease; in combination therapy with human antibodies to interleukin-18
Blood coagulation...
    disseminated intravascular; in combination therapy with human antibodies to interleukin-18
Lung,disease...
    eosinophilia; in combination therapy with human antibodies to interleukin-18
Heart,disease... Ovary,disease...
    failure; in combination therapy with human antibodies to interleukin-18
Liver,disease...
    fatty; in combination therapy with human antibodies to interleukin-18
Fertility...
    female, disorder; in combination therapy with human antibodies to interleukin-18
Lung,disease...
    fibrosis; in combination therapy with human antibodies to interleukin-18
Plasmid vectors... Drug delivery systems...
    for antibodies to human interleukin-18
Polyanhydrides... Polyesters,biological studies... Albumins,biological studies... Collagens,biological studies... Fibrins... Gelatins,biological studies... Oligosaccharides,biological studies...
Glycosaminoglycans,biological studies... Polyoxyalkylenes,biological studies...
    for delivery of antibodies to human interleukin-18
Protein sequences...
    for human antibody variable regions to interleukin-18
Escherichia coli... Saccharomyces cerevisiae...
    for preparation of antibodies to human interleukin-18
Antibodies and Immunoglobulins...
    fragments; to human interleukin-18
Kidney,disease... Inflammation...
    glomerulonephritis; in combination therapy with human antibodies to interleukin-18
Kidney,disease...
    Goodpasture's syndrome; in combination therapy with human antibodies to interleukin-18
Transplant and Transplantation...
    graft-vs.-host reaction; in combination therapy with human antibodies to interleukin-18
T cell(lymphocyte)...
    helper cell; neutralizing antibodies to human interleukin-18 modulate function of
Anemia(disease)...
    hemolytic; in combination therapy with human antibodies to interleukin-18
Purpura(disease)...
    Henoch-Schoenlein's; in combination therapy with human antibodies to interleukin-18

Infection...
    hepatitis B; in combination therapy with human antibodies to
    interleukin-18
Infection...
    hepatitis C; in combination therapy with human antibodies to
    interleukin-18
Immunotherapy...
    human antibodies to interleukin-18
Antibodies and Immunoglobulins...
    humanized; to human interleukin-18
Nervous system,disease...
    Huntington's chorea; in combination therapy with human antibodies to
    interleukin-18
Blood,disease...
    idiopathic thrombocytopenia; in combination therapy with human
    antibodies to interleukin-18
Leukocytopenia...
    idiopathic; in combination therapy with human antibodies to
    interleukin-18
Antibodies and Immunoglobulins...
    IgA, monoclonal; to human interleukin-18
Antibodies and Immunoglobulins...
    IgG1, monoclonal; to human interleukin-18
Antibodies and Immunoglobulins...
    IgG2, monoclonal; to human interleukin-18
Antibodies and Immunoglobulins...
    IgG3, monoclonal; to human interleukin-18
Antibodies and Immunoglobulins...
    IgM, monoclonal; to human interleukin-18
  CAS REGISTRY NUMBERS:
851493-81-7 851493-82-8 851493-83-9 851493-84-0 851493-46-4
    851493-47-5 851493-48-6 851493-49-7 851493-50-0 851493-51-1
    851493-52-2 851493-53-3 851493-54-4 851493-55-5 851493-56-6
    851493-57-7 851493-58-8 851493-59-9 851493-60-2 851493-61-3
    851493-62-4 851493-63-5 851493-64-6 851493-65-7 851493-66-8
    851493-67-9 851493-68-0 851493-69-1 851493-70-4 851493-71-5
    851493-72-6 851493-73-7 851493-74-8 851493-75-9 851493-76-0
    851493-77-1 851493-78-2 851493-79-3 851493-80-6 amino acid
    sequence; preparation and characterization of human antibodies to
    interleukin-18
384463-34-7 385172-72-5 391765-35-8 384440-08-8 384557-61-3
    391783-87-2 152649-01-9 391528-67-9 171212-01-4 384581-28-6
    391777-17-6 384655-19-0 389406-79-5 176145-94-1 389184-77-4
    175113-26-5 391551-76-1 391535-51-6 384735-80-2 392193-44-1
    391815-18-2 145735-36-0 184342-40-3 140025-63-4 161273-84-3
    391525-76-1 389182-06-3 140741-74-8 382730-07-6 139840-61-2
    389195-33-9 182940-44-9 150326-43-5 176348-12-2 384497-75-0
    175299-02-2 187859-73-0 384438-97-5 384442-87-9 389182-22-3
    384515-94-0 391546-79-5 391530-32-8 140062-81-3 180604-89-1
    140049-62-3 188329-74-0 391783-55-4 391847-57-7 168523-60-2
    175196-96-0 140278-85-9 156678-90-9 163953-68-2 391536-35-9
    151658-42-3 147351-39-1 150510-74-0 384728-18-1 160475-87-6
    184941-12-6 140509-06-4 392193-49-6 141010-21-1 392204-67-0
    141010-19-7 141705-88-6 391524-99-5 391766-17-9 152347-85-8
    140539-49-7 144532-01-4 172712-00-4 391528-24-8 175524-88-6
    384976-62-9 384463-75-6 391772-01-3 384463-84-7 398425-27-9
    391525-11-4 140281-74-9 140740-20-1 391527-32-5 391548-59-7
    384463-53-0 389193-51-5 164952-58-3 391550-27-9 160075-32-1
    384463-99-4 384440-42-0 384440-01-1 391524-76-8 181828-95-5
    391806-64-7 391834-40-5 140746-12-9 398114-34-6 151973-86-3
    384480-59-5 178734-25-3 384528-75-0 157417-40-8 391540-54-8

391523-44-7 168042-97-5 391523-63-0 391536-91-7 391528-57-7
391838-61-2 389357-20-4 389200-61-7 173005-29-3 183984-01-2
140033-47-2 190920-95-7 183819-07-0 384536-34-9 384506-42-7
184632-60-8 165149-73-5 384653-03-6 140098-98-2 391540-52-6
139810-14-3 384488-43-1 169733-38-4 189023-52-7 391548-28-0
384452-11-3 177929-43-0 391831-79-1 390187-88-9 392215-20-2
391535-10-7 164952-76-5 384527-97-3 391831-70-2 174128-16-6
389310-50-3 149737-37-1 391529-34-3 140576-31-4 391791-22-3
140961-11-1 391818-98-7 141165-70-0 180173-20-0 384520-60-9
189463-46-5 160900-81-2 391544-82-4 155117-74-1 140316-52-5
391557-48-5 152371-82-9 384492-73-3 391758-90-0 140030-59-7
142693-77-4 391561-76-5 391534-20-6 391532-21-1 168605-03-6
384655-55-4 391558-60-4 389415-01-4 252793-80-9 391839-12-6
150861-19-1 389406-78-4 389192-44-3 183983-99-5 186445-92-1
391547-30-1 183983-98-4 226451-68-9 398095-28-8 391532-62-0
384975-98-8 140331-48-2 183469-53-6 antibodies to human interleukin-18 for modulation of IL-18-responsive genes

9004-34-6 biological studies, for delivery of antibodies to human interleukin-18

57-50-1 biological studies, in delivery of antibodies to human interleukin-18

351881-10-2 851221-25-5 851221-27-7 851221-29-9 851221-31-3
851221-33-5 851221-35-7 384331-78-6 851221-38-0 851221-40-4
851221-42-6 851221-44-8 851221-46-0 851221-48-2 851221-49-3
851221-50-6 851221-51-7 851221-52-8 851221-53-9 851221-54-0
851221-55-1 851221-56-2 701915-28-8 851221-57-3 220541-02-6
851221-58-4 851221-59-5 851221-60-8 141977-02-8 694439-07-1
851221-61-9 706781-57-9 851221-62-0 851221-63-1 851221-64-2
851221-65-3 851221-66-4 328897-77-4 851221-67-5 851221-68-6
220540-59-0 851221-69-7 851221-70-0 851221-71-1 851221-72-2
851221-73-3 851221-74-4 851221-75-5 705288-83-1 851221-76-6
851221-77-7 851221-78-8 851221-79-9 851221-80-2 851221-81-3
851221-82-4 328897-74-1 851221-83-5 851221-84-6 851221-85-7
851221-86-8 851221-87-9 851221-88-0 851221-89-1 851221-90-4
851221-91-5 851221-92-6 851221-93-7 280106-92-5 851221-94-8
384331-70-8 851221-95-9 851221-96-0 851221-97-1 217478-43-8
851221-98-2 851221-99-3 851222-00-9 851222-01-0 848074-72-6
851222-02-1 851222-03-2 851222-04-3 851222-05-4 851222-06-5
851222-07-6 851222-08-7 173480-65-4 173480-66-5 851222-09-8 CDR regions of human antibodies to interleukin-18

14762-75-5D 15117-53-0D 10098-91-6D 14133-76-7D 15750-15-9D
14158-31-7D 10043-66-0D 14265-75-9D 13967-65-2D 15766-00-4D conjugates, uses, with antibodies to human interleukin-18

58-85-5D 10028-17-8D conjugates, with antibodies to human interleukin-18

108-31-6D copolymers with alkyl vinyl ethers, for delivery of antibodies to human interleukin-18

9004-34-6D derivs., for delivery of antibodies to human interleukin-18

9003-01-4 75268-90-5 26100-51-6 34346-01-5 26063-00-3 25248-42-4
31621-87-1 25322-68-3 40704-75-4 9002-89-5 9003-39-8 9005-32-7
9004-61-9 26023-30-3 24980-41-4 for delivery of antibodies to human interleukin-18

7585-39-9D hydroxypropyl ethers, in delivery of antibodies to human interleukin-18

59-05-2 104987-11-3 59865-13-3 53123-88-9 in combination therapy with human antibodies to interleukin-18

99-20-7 585-86-4 9004-74-4 in delivery of antibodies to human interleukin-18

691397-13-4D polyols, for delivery of antibodies to human interleukin-18

851496-80-5 unclaimed protein sequence; human antibodies to interleukin-18

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